

Refine Search

Search Results -

Terms	Documents
L10 and L9	0

Database:

US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
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Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search:

L11

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Search History

DATE: Saturday, March 04, 2006 [Printable Copy](#) [Create Case](#)

Set Name Query

side by side

DB=USPT; PLUR=YES; OP=OR

L11 L10 and l9
L10 lehmann.in.
L9 L8 and glycosylated
L8 L7 and PEG
L7 L6 and l3
L6 L5 and l4
L5 EPO and (treatment and anemia)
L4 L3 and EPO
L3 iron disturbance or deficiency
L2 L1 and (claims)
L1 (pharmaceutical composition and pharmaceutically acceptable carrier)

Hit Count Set Name

result set

0 L11
1578 L10
158 L9
234 L8
710 L7
710 L6
999 L5
3979 L4
462719 L3
3 L2
856765 L1

END OF SEARCH HISTORY

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NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	DEC 05	CASREACT(R) - Over 10 million reactions available
NEWS	4	DEC 14	2006 MeSH terms loaded in MEDLINE/LMEDLINE
NEWS	5	DEC 14	2006 MeSH terms loaded for MEDLINE file segment of TOXCENTER
NEWS	6	DEC 14	CA/Caplus to be enhanced with updated IPC codes
NEWS	7	DEC 21	IPC search and display fields enhanced in CA/Caplus with the IPC reform
NEWS	8	DEC 23	New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/USPAT2
NEWS	9	JAN 13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS	10	JAN 13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
NEWS	11	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS	12	JAN 17	IPC 8 in the WPI family of databases including WPIFV
NEWS	13	JAN 30	Saved answer limit increased
NEWS	14	JAN 31	Monthly current-awareness alert (SDI) frequency added to TULSA
NEWS	15	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS	16	FEB 22	Status of current WO (PCT) information on STN
NEWS	17	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS	18	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS	19	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS	20	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS	21	FEB 28	TOXCENTER reloaded with enhancements
NEWS	22	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS	23	MAR 01	INSPEC reloaded and enhanced
NEWS	24	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS EXPRESS			FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005. V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT http://download.cas.org/express/v8.0-Discover/
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NEWS INTER			General Internet Information
NEWS LOGIN			Welcome Banner and News Items
NEWS PHONE			Direct Dial and Telecommunication Network Access to STN
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FILE 'HOME' ENTERED AT 09:26:32 ON 04 MAR 2006

=> file medline, uspatful, dgene, embase,wpids, fsta, jicst, biosis, scisearch		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	1.26	1.26

FILE 'MEDLINE' ENTERED AT 09:30:19 ON 04 MAR 2006

FILE 'USPATFULL' ENTERED AT 09:30:19 ON 04 MAR 2006
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=> s erythropoietin rpotein
L1 0 ERYTHROPOIETIN RPOTEIN

=> s EPO or erythropoietin protein
L2 61560 EPO OR ERYTHROPOIETIN PROTEIN

=> s l2 and darbepoetin
L3 228 L2 AND DARBEPOETIN

=> s l3 and alfa
L4 169 L3 AND ALFA

=> s l2 and (epoetin beta or alfa)
L5 1068 L2 AND (EPOETIN BETA OR ALFA)

=> s l5 and l4
L6 169 L5 AND L4

=> s l2 and (PEG or pegylated)
L7 3670 L2 AND (PEG OR PEGYLATED)

=> s l7 and l6
L8 36 L7 AND L6

=> s 18 and treatment
L9 34 L8 AND TREATMENT

=> d 19 ti abs ibib 1-10

L9 ANSWER 1 OF 34 USPATFULL on STN
TI Stable suspension formulations of erythropoietin receptor agonists
AB A suspension formulation for therapeutic use includes a non-aqueous, single-phase vehicle exhibiting viscous fluid characteristics and a particle formulation comprising an erythropoietin receptor agonist dispersed in the vehicle.

ACCESSION NUMBER: 2006:34752 USPATFULL
TITLE: Stable suspension formulations of erythropoietin receptor agonists
INVENTOR(S): Liu, Kui, Redwood City, CA, UNITED STATES
Desjardin, Michael A., Sunnyvale, CA, UNITED STATES
Hill, Beth L., Sunnyvale, CA, UNITED STATES
Li, Zengji, San Ramon, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006030526	A1	20060209
APPLICATION INFO.:	US 2005-194850	A1	20050801 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-599564P	20040805 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	DEWIPAT INCORPORATED, 4606 FM 1960 WEST, SUITE 400, PMB 166, HOUSTON, TX, 77069, US	
NUMBER OF CLAIMS:	20	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	3 Drawing Page(s)	
LINE COUNT:	618	

L9 ANSWER 2 OF 34 USPATFULL on STN
TI Stable particle formulations of erythropoietin receptor agonists
AB A particle formulation includes an erythropoietin receptor agonist, a buffer, and a sugar, wherein the buffer and sugar stabilize the erythropoietin receptor agonist against aggregation.

ACCESSION NUMBER: 2006:33784 USPATFULL
TITLE: Stable particle formulations of erythropoietin receptor agonists
INVENTOR(S): Liu, Kui, Redwood City, CA, UNITED STATES
Desjardin, Michael A., Sunnyvale, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006029551	A1	20060209
APPLICATION INFO.:	US 2005-194889	A1	20050801 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-599663P	20040805 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	DEWIPAT INCORPORATED, 4606 FM 1960 WEST, SUITE 400, PMB 166, HOUSTON, TX, 77069, US	
NUMBER OF CLAIMS:	20	

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 603

L9 ANSWER 3 OF 34 USPATFULL on STN

TI Use of erythropoietin for **treatment** of cancer

AB A method for treating cancer is provided. The method involves administering to subjects in need of such **treatment** an effective amount of erythropoietin of an analog thereof effective to inhibit angiogenesis in a tumor. Also provided are methods to reduce HIF-1 α protein levels and/or VEGF expression, particularly in tumors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:306385 USPATFULL

TITLE: Use of erythropoietin for **treatment** of cancer

INVENTOR(S): Lounsbury, Karen M., Essex Junction, VT, UNITED STATES
Wong, Cheung, South Burlington, VT, UNITED STATES
Hale, Sarah A., Essex Junction, VT, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005267027	A1	20051201
APPLICATION INFO.:	US 2005-93177	A1	20050328 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-559479P	20040405 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	WOLF GREENFIELD & SACKS, PC, FEDERAL RESERVE PLAZA, 600 ATLANTIC AVENUE, BOSTON, MA, 02210-2211, US	
NUMBER OF CLAIMS:	23	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	4 Drawing Page(s)	
LINE COUNT:	1151	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 4 OF 34 USPATFULL on STN

TI Combination dosing regimen for erythropoietin

AB The present invention provides a combination dosing regimen for erythropoietin (**EPO**). More particularly, the present dosing regimen includes administration of at least a first dosing segment comprising a first exposure to **EPO** capable of stimulating the production of reticulocytes followed by a second exposure to **EPO** capable of sustaining the maturation of the reticulocytes into neocytes, and ultimately, red blood cells. Advantageously, the dosing segment may be cycled or repeated, any number of times and according to any desired time scheme, in order to provide or maintain any desired total red blood cell count and/or hemoglobin concentration. Methods of **treatment** employing the combination dosing regimen, as well as kits are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:306384 USPATFULL

TITLE: Combination dosing regimen for erythropoietin

INVENTOR(S): Cheung, Wing K., Warren, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005267026	A1	20051201
APPLICATION INFO.:	US 2005-88284	A1	20050324 (11)

	NUMBER	DATE
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PRIORITY INFORMATION:	US 2004-556923P	20040326 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	PHILIP S. JOHNSON, JOHNSON & JOHNSON, ONE JOHNSON & JOHNSON PLAZA, NEW BRUNSWICK, NJ, 08933-7003, US	
NUMBER OF CLAIMS:	40	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	22 Drawing Page(s)	
LINE COUNT:	1077	
CAS INDEXING IS AVAILABLE FOR THIS PATENT.		

L9 ANSWER 5 OF 34 USPATFULL on STN

TI Albumin fusion proteins

AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2005:305894 USPATFULL
TITLE:	Albumin fusion proteins
INVENTOR(S):	Ballance, David J., Berwyn, PA, UNITED STATES Sleep, Darrell, West Bridgford, UNITED KINGDOM Prior, Christopher P., Rosemont, PA, UNITED STATES Sadeghi, Homayoun, Doylestown, PA, UNITED STATES Turner, Andrew J., Eagleville, PA, UNITED STATES
PATENT ASSIGNEE(S):	Human Genome Sciences, Inc. (U.S. corporation) Delta Biotechnology Limited (U.S. corporation)

	NUMBER	KIND	DATE
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PATENT INFORMATION:	US 2005266533	A1	20051201
APPLICATION INFO.:	US 2005-78914	A1	20050314 (11)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2001-832501, filed on 12 Apr 2001, ABANDONED		

	NUMBER	DATE
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PRIORITY INFORMATION:	US 2000-256931P	20001221 (60)
	US 2000-199384P	20000425 (60)
	US 2000-229358P	20000412 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	FINNEGAN, HENDERSON, FARABOW, GARRETT & DUNNER, LLP, 901 NEW YORK AVENUE, NW, WASHINGTON, DC, 20001-4413, US	
NUMBER OF CLAIMS:	21	
EXEMPLARY CLAIM:	1-60	
NUMBER OF DRAWINGS:	20 Drawing Page(s)	
LINE COUNT:	13941	
CAS INDEXING IS AVAILABLE FOR THIS PATENT.		

L9 ANSWER 6 OF 34 USPATFULL on STN

TI **Treatment** of neurological dysfunction comprising fructopyranose sulfamates and erythropoietin

AB Co-therapy for the **treatment** of neurological dysfunctions

comprising administration of one or more fructopyranose sulfamates and erythropoietin.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:299513 USPATFULL
TITLE: **Treatment** of neurological dysfunction
comprising fructopyranose sulfamates and erythropoietin
INVENTOR(S): Plata-Salaman, Carlos R., Zionsville, IN, UNITED STATES
Smith-Swintosky, Virginia, Hatfield, PA, UNITED STATES
PATENT ASSIGNEE(S): Ortho-McNeil Pharmaceutical, Inc., Raritan, NJ, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005261182	A1	20051124
APPLICATION INFO.:	US 2005-47420	A1	20050131 (11)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2002-56828, filed on 24 Jan 2002, GRANTED, Pat. No. US 6908902		

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-266194P	20010202 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	WOODCOCK WASHBURN LLP, ONE LIBERTY PLACE, 46TH FLOOR, PHILADELPHIA, PA, 19103, US	
NUMBER OF CLAIMS:	20	
EXEMPLARY CLAIM:	1	
LINE COUNT:	1198	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 7 OF 34 USPATFULL on STN
TI Immunoglobulin chimeric monomer-dimer hybrids
AB The invention relates to a chimeric monomer-dimer hybrid protein wherein said protein comprises a first and a second polypeptide chain, said first polypeptide chain comprising at least a portion of an immunoglobulin constant region and a biologically active molecule, and said second polypeptide chain comprising at least a portion of an immunoglobulin constant region without the biologically active molecule of the first chain. The invention also relates to methods of using and methods of making the chimeric monomer-dimer hybrid protein of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:298530 USPATFULL
TITLE: Immunoglobulin chimeric monomer-dimer hybrids
INVENTOR(S): Peters, Robert T., West Roxbury, MA, UNITED STATES
Mezo, Adam R., Waltham, MA, UNITED STATES
Rivera, Daniel S., Providence, RI, UNITED STATES
Bitonti, Alan J., Acton, MA, UNITED STATES
Low, Susan C., Pepperell, MA, UNITED STATES
Stattel, James, Leominster, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005260194	A1	20051124
APPLICATION INFO.:	US 2005-29003	A1	20050105 (11)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2004-841250, filed on 6 May 2004, PENDING		

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-469600P	20030506 (60)

US 2003-487964P 20030717 (60)
 US 2004-539207P 20040126 (60)
 DOCUMENT TYPE: Utility
 FILE SEGMENT: APPLICATION
 LEGAL REPRESENTATIVE: FINNEGAN, HENDERSON, FARABOW, GARRETT & DUNNER, LLP,
 901 NEW YORK AVENUE, NW, WASHINGTON, DC, 20001-4413, US
 NUMBER OF CLAIMS: 131
 EXEMPLARY CLAIM: 1
 NUMBER OF DRAWINGS: 27 Drawing Page(s)
 LINE COUNT: 5395
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 8 OF 34 USPATFULL on STN
 TI Genomic modification
 AB The invention includes transchromosomal avians and transchromosomal
 avian cells and methods for the introduction of artificial chromosomes
 into the genome of avians and avian cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
 ACCESSION NUMBER: 2005:228844 USPATFULL
 TITLE: Genomic modification
 INVENTOR(S): Christmann, Leandro, Watkinsville, GA, UNITED STATES
 Eberhardt, Dawn M., Danielsville, GA, UNITED STATES
 Harvey, Alex J., Athens, GA, UNITED STATES
 Leavitt, Markley C., Watkinsville, GA, UNITED STATES
 PATENT ASSIGNEE(S): AviGenics, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005198700	A1	20050908
APPLICATION INFO.:	US 2005-68155	A1	20050228 (11)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2004-940315, filed on 14 Sep 2004, PENDING Continuation-in-part of Ser. No. US 2004-811136, filed on 26 Mar 2004, PENDING Continuation-in-part of Ser. No. US 2004-790455, filed on 1 Mar 2004, PENDING		

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-453126P	20030307 (60)
	US 2003-490452P	20030728 (60)
	US 2004-536677P	20040115 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	AVIGENICS, INC., 111 RIVERBEND ROAD, ATHENS, GA, 30605, US	
NUMBER OF CLAIMS:	33	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	43 Drawing Page(s)	
LINE COUNT:	5542	
CAS INDEXING IS AVAILABLE FOR THIS PATENT.		

L9 ANSWER 9 OF 34 USPATFULL on STN
 TI Albumin fusion proteins
 AB The present invention encompasses albumin fusion proteins. Nucleic acid
 molecules encoding the albumin fusion proteins of the invention are also
 encompassed by the invention, as are vectors containing these nucleic
 acids, host cells transformed with these nucleic acids vectors, and
 methods of making the albumin fusion proteins of the invention and using
 these nucleic acids, vectors, and/or host cells. Additionally the
 present invention encompasses pharmaceutical compositions comprising
 albumin fusion proteins and methods of treating, preventing, or
 ameliorating diseases, disorders or conditions using albumin fusion

proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:214989 USPATFULL
TITLE: Albumin fusion proteins
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Haseltine, William A., Washington, DC, UNITED STATES
Ballance, David J., Berwyn, PA, UNITED STATES
Turner, Andrew J., Eagleville, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005186664	A1	20050825
APPLICATION INFO.:	US 2004-775204	A1	20040211 (10)
RELATED APPLN. INFO.:	Continuation of Ser. No. WO 2002-US40891, filed on 23 Dec 2002, PENDING		

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-341811P	20011221 (60)
	US 2002-350358P	20020124 (60)
	US 2002-351360P	20020128 (60)
	US 2002-359370P	20020226 (60)
	US 2002-360000P	20020228 (60)
	US 2002-367500P	20020327 (60)
	US 2002-370227P	20020408 (60)
	US 2002-378950P	20020510 (60)
	US 2002-382617P	20020524 (60)
	US 2002-383123P	20020528 (60)
	US 2002-385708P	20020605 (60)
	US 2002-394625P	20020710 (60)
	US 2002-398008P	20020724 (60)
	US 2002-402131P	20020809 (60)
	US 2002-402708P	20020813 (60)
	US 2002-411355P	20020918 (60)
	US 2002-411426P	20020918 (60)
	US 2002-414984P	20021002 (60)
	US 2002-417611P	20021011 (60)
	US 2002-420246P	20021023 (60)
	US 2002-423623P	20021105 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, INTELLECTUAL PROPERTY DEPT.,
14200 SHADY GROVE ROAD, ROCKVILLE, MD, 20850, US

NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 23 Drawing Page(s)
LINE COUNT: 25129
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 10 OF 34 USPATFULL on STN
TI Method of treating disturbances of iron distribution in inflammatory
intestinal diseases
AB The present invention relates to the use of erythropoietin for the
treatment of disturbances of iron distribution in chronic
inflammatory intestinal diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:209503 USPATFULL
TITLE: Method of treating disturbances of iron distribution in
inflammatory intestinal diseases
INVENTOR(S): Klima, Horst, Penzberg, GERMANY, FEDERAL REPUBLIC OF
Lehmann, Paul, Worms, GERMANY, FEDERAL REPUBLIC OF

Roeddiger, Ralf, Gorxheimertal, GERMANY, FEDERAL
REPUBLIC OF
Walter-Matsui, Ruth, Altenbuseck, GERMANY, FEDERAL
REPUBLIC OF

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005181986	A1	20050818
APPLICATION INFO.:	US 2004-13560	A1	20041216 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	EP 2003-104832	20031219
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	HOFFMANN-LA ROCHE INC., PATENT LAW DEPARTMENT, 340 KINGSLAND STREET, NUTLEY, NJ, 07110, US	
NUMBER OF CLAIMS:	15	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	1 Drawing Page(s)	
LINE COUNT:	820	
CAS INDEXING IS AVAILABLE FOR THIS PATENT.		

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(FILE 'HOME' ENTERED AT 09:26:32 ON 04 MAR 2006)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JICST-EPLUS,
BIOSIS, SCISEARCH' ENTERED AT 09:30:19 ON 04 MAR 2006

L1 0 S ERYTHROPOIETIN RPOTEIN
L2 61560 S EPO OR ERYTHROPOIETIN PROTEIN
L3 228 S L2 AND DARBEPOETIN
L4 169 S L3 AND ALFA
L5 1068 S L2 AND (EPOETIN BETA OR ALFA)
L6 169 S L5 AND L4
L7 3670 S L2 AND (PEG OR PEGYLATED)
L8 36 S L7 AND L6
L9 34 S L8 AND TREATMENT

=> e lehmann, p/au

E1 1 LEHMANN ZIEBARTH N/AU
E2 1 LEHMANN ZIEBARTH NICOLAS/AU
E3 0 --> LEHMANN, P/AU
E4 1 LEHMANNB E/AU
E5 1 LEHMANNBAERTS M/AU
E6 1 LEHMANNBEI G/AU
E7 1 LEHMANNBODEM C/AU
E8 1 LEHMANNBRAUNS S/AU
E9 1 LEHMANNBROCKHAUS E/AU
E10 9 LEHMANNBRUINSMA K/AU
E11 4 LEHMANNBURGEL U/AU
E12 1 LEHMANNBURI T/AU

=> d his

(FILE 'HOME' ENTERED AT 09:26:32 ON 04 MAR 2006)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, JICST-EPLUS,
BIOSIS, SCISEARCH' ENTERED AT 09:30:19 ON 04 MAR 2006

L1 0 S ERYTHROPOIETIN RPOTEIN
L2 61560 S EPO OR ERYTHROPOIETIN PROTEIN
L3 228 S L2 AND DARBEPOETIN

L4 169 S L3 AND ALFA
 L5 1068 S L2 AND (EPOETIN BETA OR ALFA)
 L6 169 S L5 AND L4
 L7 3670 S L2 AND (PEG OR PEGYLATED)
 L8 36 S L7 AND L6
 L9 34 S L8 AND TREATMENT
 E LEHMANN, P/AU

=> s l2 and conjugate
 L10 3248 L2 AND CONJUGATE

=> s l10 and mutatn
 L11 0 L10 AND MUTATN

=> s l10 and mutant
 L12 1886 L10 AND MUTANT

=> s l12 and (glycosylation0
 UNMATCHED LEFT PARENTHESIS 'AND (GLYCOSYLAT'
 The number of right parentheses in a query must be equal to the
 number of left parentheses.

=> s l12 and (glycosylation)
 L13 1273 L12 AND (GLYCOSYLATION)

=> s l13 and l9
 L14 8 L13 AND L9

=> d l14 ti abs ibib tot

L14 ANSWER 1 OF 8 USPATFULL on STN
 TI Stable suspension formulations of erythropoietin receptor agonists
 AB A suspension formulation for therapeutic use includes a non-aqueous,
 single-phase vehicle exhibiting viscous fluid characteristics and a
 particle formulation comprising an erythropoietin receptor agonist
 dispersed in the vehicle.

ACCESSION NUMBER: 2006:34752 USPATFULL
 TITLE: Stable suspension formulations of erythropoietin
 receptor agonists
 INVENTOR(S): Liu, Kui, Redwood City, CA, UNITED STATES
 Desjardin, Michael A., Sunnyvale, CA, UNITED STATES
 Hill, Beth L., Sunnyvale, CA, UNITED STATES
 Li, Zengji, San Ramon, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006030526	A1	20060209
APPLICATION INFO.:	US 2005-194850	A1	20050801 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-599564P	20040805 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	DEWIPAT INCORPORATED, 4606 FM 1960 WEST, SUITE 400, PMB 166, HOUSTON, TX, 77069, US	
NUMBER OF CLAIMS:	20	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	3 Drawing Page(s)	
LINE COUNT:	618	

L14 ANSWER 2 OF 8 USPATFULL on STN

TI Stable particle formulations of erythropoietin receptor agonists
AB A particle formulation includes an erythropoietin receptor agonist, a buffer, and a sugar, wherein the buffer and sugar stabilize the erythropoietin receptor agonist against aggregation.

ACCESSION NUMBER: 2006:33784 USPATFULL
TITLE: Stable particle formulations of erythropoietin receptor agonists
INVENTOR(S): Liu, Kui, Redwood City, CA, UNITED STATES
Desjardin, Michael A., Sunnyvale, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006029551	A1	20060209
APPLICATION INFO.:	US 2005-194889	A1	20050801 (11)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-599663P	20040805 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	DEWIPAT INCORPORATED, 4606 FM 1960 WEST, SUITE 400, PMB 166, HOUSTON, TX, 77069, US	
NUMBER OF CLAIMS:	20	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	7 Drawing Page(s)	
LINE COUNT:	603	

L14 ANSWER 3 OF 8 USPATFULL on STN

TI Albumin fusion proteins
AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:305894 USPATFULL
TITLE: Albumin fusion proteins
INVENTOR(S): Ballance, David J., Berwyn, PA, UNITED STATES
Sleep, Darrell, West Bridgford, UNITED KINGDOM
Prior, Christopher P., Rosemont, PA, UNITED STATES
Sadeghi, Homayoun, Doylestown, PA, UNITED STATES
Turner, Andrew J., Eagleville, PA, UNITED STATES
PATENT ASSIGNEE(S): Human Genome Sciences, Inc. (U.S. corporation)
Delta Biotechnology Limited (U.S. corporation).

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005266533	A1	20051201
APPLICATION INFO.:	US 2005-78914	A1	20050314 (11)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2001-832501, filed on 12 Apr 2001, ABANDONED		

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-256931P	20001221 (60)
	US 2000-199384P	20000425 (60)

US 2000-229358P 20000412 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FINNEGAN, HENDERSON, FARABOW, GARRETT & DUNNER, LLP,
901 NEW YORK AVENUE, NW, WASHINGTON, DC, 20001-4413, US
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1-60
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 13941
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 4 OF 8 USPATFULL on STN

TI Immunoglobulin chimeric monomer-dimer hybrids
AB The invention relates to a chimeric monomer-dimer hybrid protein wherein said protein comprises a first and a second polypeptide chain, said first polypeptide chain comprising at least a portion of an immunoglobulin constant region and a biologically active molecule, and said second polypeptide chain comprising at least a portion of an immunoglobulin constant region without the biologically active molecule of the first chain. The invention also relates to methods of using and methods of making the chimeric monomer-dimer hybrid protein of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:298530 USPATFULL
TITLE: Immunoglobulin chimeric monomer-dimer hybrids
INVENTOR(S): Peters, Robert T., West Roxbury, MA, UNITED STATES
Mezo, Adam R., Waltham, MA, UNITED STATES
Rivera, Daniel S., Providence, RI, UNITED STATES
Bitonti, Alan J., Acton, MA, UNITED STATES
Low, Susan C., Pepperell, MA, UNITED STATES
Stattel, James, Leominster, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005260194	A1	20051124
APPLICATION INFO.:	US 2005-29003	A1	20050105 (11)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2004-841250, filed on 6 May 2004, PENDING		

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-469600P	20030506 (60)
	US 2003-487964P	20030717 (60)
	US 2004-539207P	20040126 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FINNEGAN, HENDERSON, FARABOW, GARRETT & DUNNER, LLP,
901 NEW YORK AVENUE, NW, WASHINGTON, DC, 20001-4413, US
NUMBER OF CLAIMS: 131
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Page(s)
LINE COUNT: 5395
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 5 OF 8 USPATFULL on STN

TI Albumin fusion proteins
AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the

present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:214989 USPATFULL
TITLE: Albumin fusion proteins
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Haseltine, William A., Washington, DC, UNITED STATES
Ballance, David J., Berwyn, PA, UNITED STATES
Turner, Andrew J., Eagleville, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005186664	A1	20050825
APPLICATION INFO.:	US 2004-775204	A1	20040211 (10)
RELATED APPLN. INFO.:	Continuation of Ser. No. WO 2002-US40891, filed on 23 Dec 2002, PENDING		

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-341811P	20011221 (60)
	US 2002-350358P	20020124 (60)
	US 2002-351360P	20020128 (60)
	US 2002-359370P	20020226 (60)
	US 2002-360000P	20020228 (60)
	US 2002-367500P	20020327 (60)
	US 2002-370227P	20020408 (60)
	US 2002-378950P	20020510 (60)
	US 2002-382617P	20020524 (60)
	US 2002-383123P	20020528 (60)
	US 2002-385708P	20020605 (60)
	US 2002-394625P	20020710 (60)
	US 2002-398008P	20020724 (60)
	US 2002-402131P	20020809 (60)
	US 2002-402708P	20020813 (60)
	US 2002-411355P	20020918 (60)
	US 2002-411426P	20020918 (60)
	US 2002-414984P	20021002 (60)
	US 2002-417611P	20021011 (60)
	US 2002-420246P	20021023 (60)
	US 2002-423623P	20021105 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, INTELLECTUAL PROPERTY DEPT.,
14200 SHADY GROVE ROAD, ROCKVILLE, MD, 20850, US
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 23 Drawing Page(s)
LINE COUNT: 25129
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 6 OF 8 USPATFULL on STN

TI Methods of using Flt3-Ligand in hematopoietic cell transplantation procedures incorporating nonmyeloablative conditioning regimens
AB The invention is directed to methods of using Flt3-Ligand in hematopoietic cell transplantation procedures using nonmyeloablative conditioning regimens. This abstract is provided for the sole purpose of enabling the reader to quickly ascertain the subject matter of the technical disclosure and is not intended to be used to interpret or limit the scope or meaning of the claims.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:68484 USPATFULL
TITLE: Methods of using Flt3-Ligand in hematopoietic cell
transplantation procedures incorporating
nonmyeloablative conditioning regimens
INVENTOR(S): Lyman, Stewart D., Seattle, WA, UNITED STATES
Beckmann, M. Patricia, Hansville, WA, UNITED STATES
McKenna, Hilary J., Seattle, WA, UNITED STATES
Nash, Richard A., Seattle, WA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005058622	A1	20050317
APPLICATION INFO.:	US 2003-730334	A1	20031208 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-431266P	20021206 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	Immunex Corporation, Law Department, 1201 Amgen Court West, Seattle, WA, 98119	
NUMBER OF CLAIMS:	25	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	4 Drawing Page(s)	
LINE COUNT:	3259	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 7 OF 8 USPATFULL on STN

TI Immunoglobulin chimeric monomer-dimer hybrids
AB The invention relates to a chimeric monomer-dimer hybrid protein wherein
said protein comprises a first and a second polypeptide chain, said
first polypeptide chain comprising at least a portion of an
immunoglobulin constant region and a biologically active molecule, and
said second polypeptide chain comprising at least a portion of an
immunoglobulin constant region without the biologically active molecule
of the first chain. The invention also relates to methods of using and
methods of making the chimeric monomer-dimer hybrid protein of the
invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2005:37495 USPATFULL
TITLE: Immunoglobulin chimeric monomer-dimer hybrids
INVENTOR(S): Peters, Robert T., West Roxbury, MA, UNITED STATES
Mezo, Adam R., Waltham, MA, UNITED STATES
Rivera, Daniel S., Providence, RI, UNITED STATES
Bitonti, Alan J., Acton, MA, UNITED STATES
Stattel, James, Leominster, MA, UNITED STATES
Low, Susan C., Pepperell, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005032174	A1	20050210
APPLICATION INFO.:	US 2004-841250	A1	20040506 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-487964P	20030717 (60)
	US 2004-539207P	20040126 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	Finnegan, Henderson, Farabow,, Garrett & Dunner, L.L.P., 1300 I Street, N.W., Washington, DC, 20005-3315	

NUMBER OF CLAIMS: 154
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Page(s)
LINE COUNT: 5512
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 8 OF 8 USPATFULL on STN

TI Albumin fusion proteins

AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2003:282700 USPATFULL
TITLE: Albumin fusion proteins
INVENTOR(S): Ballance, David J., Berwyn, PA, UNITED STATES
Sleep, Darrell, West Bridgford, UNITED KINGDOM
Prior, Christopher P., Rosemont, PA, UNITED STATES
Sadeghi, Homayoun, Doylestown, PA, UNITED STATES
Turner, Andrew J., Eagleville, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003199043	A1	20031023
APPLICATION INFO.:	US 2001-832501	A1	20010412 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-256931P	20001221 (60)
	US 2000-199384P	20000425 (60)
	US 2000-229358P	20000412 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850	
NUMBER OF CLAIMS:	60	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	18 Drawing Page(s)	
LINE COUNT:	14339	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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NEWS	5	DEC 14	2006 MeSH terms loaded for MEDLINE file segment of TOXCENTER
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NEWS	7	DEC 21	IPC search and display fields enhanced in CA/CAPLUS with the IPC reform
NEWS	8	DEC 23	New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/USPAT2
NEWS	9	JAN 13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS	10	JAN 13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
NEWS	11	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS	12	JAN 17	IPC 8 in the WPI family of databases including WPIFV.
NEWS	13	JAN 30	Saved answer limit increased
NEWS	14	JAN 31	Monthly current-awareness alert (SDI) frequency added to TULSA
NEWS	15	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS	16	FEB 22	Status of current WO (PCT) information on STN
NEWS	17	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS	18	FEB 22	Updates in EPPFULL; IPC 8 enhancements added
NEWS	19	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS	20	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS	21	FEB 28	TOXCENTER reloaded with enhancements
NEWS	22	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS	23	MAR 01	INSPEC reloaded and enhanced
NEWS	24	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS EXPRESS			FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005. V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT http://download.cas.org/express/v8.0-Discover/
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=> file medline

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 09:37:26 ON 04 MAR 2006

FILE LAST UPDATED: 3 MAR 2006 (20060303/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>).
See also:

<http://www.nlm.nih.gov/mesh/>
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s EPO and heart disease

5328 EPO
703771 HEART
1753541 DISEASE
89451 HEART DISEASE
(HEART(W)DISEASE)

L1 19 EPO AND HEART DISEASE

=> s EPO and anemia

5328 EPO
103744 ANEMIA

L2 1544 EPO AND ANEMIA

=> s l2 and l1

L3 5 L2 AND L1

=> d l3 ti abs ibib tot

L3 ANSWER 1 OF 5 MEDLINE on STN

TI Erythropoietin in cardiovascular diseases.

AB Several studies showed that anaemia is commonly observed in patients with Chronic Heart Failure (CHF) and is associated with worsened symptoms and survival. When anaemia in these patients is treated with erythropoietin (EPO), a significant improvement in cardiac function and symptoms was observed. Although it was originally believed that EPO specifically acted on haematopoietical cells, recent evidence demonstrated

several non-haematopoietical effects. Ischaemia/reperfusion experiments in rat heart and brain showed large infarct reduction when treated with **EPO**. Other effects of **EPO** are related to its pro-angiogenic effects on endothelial cells, which could be of potential value in patients with ischaemic **heart disease**. These preclinical findings suggest that **EPO** may have potential effects in cardiovascular disease beyond correction of haemoglobin levels.

ACCESSION NUMBER: 2004095360 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14984916
TITLE: Erythropoietin in cardiovascular diseases.
AUTHOR: van der Meer Peter; Voors Adriaan A; Lipsic Erik; van Gilst Wiek H; van Veldhuisen Dirk J
CORPORATE SOURCE: Department of Cardiology, University Hospital Groningen Groningen, The Netherlands.
SOURCE: European heart journal, (2004 Feb) Vol. 25, No. 4, pp. 285-91. Ref: 63
Journal code: 8006263. ISSN: 0195-668X.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200406
ENTRY DATE: Entered STN: 20040302
Last Updated on STN: 20040602
Entered Medline: 20040601

L3 ANSWER 2 OF 5 MEDLINE on STN

TI Homocysteine and C-reactive protein levels in haemodialysis patients.

AB BACKGROUND: Mild to moderate hyperhomocysteinemia is very common among patients undergoing haemodialysis. There is sufficient evidence that hyperhomocysteinemia is an independent risk factor for cardiovascular and or atheromatous disease in end stage renal failure patients. Vitamin supplementation such as vitamin B6, B12 or folate has been proposed to correct this metabolic disturbance and it is to be proved if this intervention benefit these patients, but there is no agreement whether oral folate supplementation is capable to normalize homocysteine levels in end stage renal failure patients. METHODS: In 53 patients, undergoing haemodialysis, homocysteine levels (Hcy), folate, vitamin B12, ferritin and C-reactive protein (CRP) were estimated before and after dialysis, without folate supplementation. Thirty voluntary blood donors were used as controls to compare homocysteine levels. After four weeks of oral folate supplementation (10 mg/24 hours) the levels of homocysteine, serum folate and intra-erythrocyte folate were estimated again. Eighteen months later the survival rate of our patients was recorded and analyzed in relation to Hcy and CRP levels. RESULTS: The results showed that haemodialysis patients exhibited, almost, fourfold higher homocysteine levels than controls (27.39 +/- 11.54 vs 7.38 +/- 3.5, $t = -8.2$, $p = 0.000000$). Folate levels, vitamin B12 and CRP increase significantly after haemodialysis where as homocysteine levels decrease (Hcy1 vs. Hcy2: $z = 2.08$, $p = 0.03$). Fourteen (14) patients suffered from coronary **heart disease** (CHD) and they exhibited the higher levels of homocysteine (Hcy1 vs. CHD: $z = -3.4$, $p = 0.0006$). All estimations performed revealed a negative correlation between homocysteine levels and plasma or intra-erythrocyte folate. No other variable exhibited any significant influence upon homocysteine levels. After folate supplementation homocysteine levels in the whole number of patients were unchanged (Hcy(before) vs. Hcy(after): 27.39 +/- 11.54 vs. 26.95 +/- 8.22, $z = 0.3$, $p = 0.7$, NS). When patients with homocysteine levels higher than 24 micromol/L were selected, a significant decrease was observed (34.77 +/- 9.32 vs. 30.0 +/- 8.05, $z = 2.09$, $p = 0.02$). Forty-two patients were treated with erythropoietin for their **anemia** and we found a positive correlation between C-reactive

protein levels and rhu-Epo dose (CRP vs. Epo: $r = 0.45$, $p = 0.002$). Homocysteine levels did not exhibit any significant influence upon short-term survival ($U = -0.37$, $p = 0.3$, NS) whereas CRP levels exhibit a significant influence upon short-term survival ($U = 2.15$, $p = 0.005$). CONCLUSIONS: Homocysteine levels in haemodialysis patients are fourfold higher than healthy controls. Folate, vitamin B12 and CRP increases significantly after dialysis. Patients with coronary **heart disease** exhibit the highest levels of homocysteine. The homocysteine levels are inversely related with the folate levels. The exogenous folate supplementation increase the serum folate levels but decreases homocysteine only in patients with higher than mild hyperhomocysteinemia. Hcy doesn't exert any significant effect upon the short-term survival of the haemodialysis patients but CRP level is a good predictor of the short-term survival of these patients.

ACCESSION NUMBER: 2002349838 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12092633
TITLE: Homocysteine and C-reactive protein levels in haemodialysis patients.
AUTHOR: Koulouridis E; Tzilianos M; Katsarou A; Costimba I; Klonou E; Panagiotaki E; Georgalidis C; Krokida A; Delaportas N; Lachanas A; Karaliotas G; Kaliolia I
CORPORATE SOURCE: Nephrology Department, General Hospital of Corfu, Greece.. koulef@otenet.gr
SOURCE: International urology and nephrology, (2001) Vol. 33, No. 2, pp. 207-15.
Journal code: 0262521. ISSN: 0301-1623.
PUB. COUNTRY: Hungary
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 20020703
Last Updated on STN: 20021220
Entered Medline: 20021219

L3 ANSWER 3 OF 5 MEDLINE on STN

TI A rationale for an individualized haemoglobin target.

AB Despite the use of recombinant human erythropoietin (rh-EPO, epoetin) for more than a decade in treating renal anaemia, there is still considerable debate over optimal target haemoglobin (Hb) levels. Current European and North American guidelines that are based on decade-old trials aim for partial anaemia correction, with a subnormal target Hb concentration. More recent randomized clinical trials examining the effect of normalizing Hb levels have produced conflicting results. A study in the USA, in patients with existing congestive heart failure or ischaemic **heart disease**, showed an unexpected rise in cardiac mortality and haemodialysis access failure with higher Hb levels. In contrast, three other studies (in Australia, Spain and Canada) that normalized Hb levels in healthier dialysis patients observed improvements in quality of life and exercise capacity and a slower progression of left ventricular dilatation, without an unacceptable increase in the incidence of adverse effects. These studies indicate that, while higher Hb levels may be detrimental to patients with pre-existing cardiac disease, healthier patients benefit from normalized Hb levels. Thus, there is no clear scientific rationale for setting a single Hb target for all patients, and individualized treatment targets would appear to be a more logical and patient-centred approach.

ACCESSION NUMBER: 2002348738 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12091594
TITLE: A rationale for an individualized haemoglobin target.
AUTHOR: Muirhead Norman
CORPORATE SOURCE: University of Western Ontario, London, Ontario, Canada.
SOURCE: Nephrology, dialysis, transplantation : official

publication of the European Dialysis and Transplant
Association - European Renal Association, (2002) Vol. 17
Suppl 6, pp. 2-7. Ref: 23
Journal code: 8706402. ISSN: 0931-0509.

PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20020702
Last Updated on STN: 20030214
Entered Medline: 20030212

L3 ANSWER 4 OF 5 MEDLINE on STN

TI Anaemia in chronic renal disease: lessons learned since Seville 1994.

AB Cardiovascular disease is the major cause of death among patients with end-stage renal disease, accounting for almost half of all fatalities. In recent years much progress has been made in understanding the pathogenesis of cardiovascular disease in the uraemic population. Anaemia is a consistent finding in chronic renal disease, affecting up to 90% of patients, and the central role of anaemia in the development of cardiovascular dysfunction is now well established. A significant proportion of patients have established cardiovascular complications on initiation of dialysis, raising the possibility of early correction of anaemia as a strategy for preventing cardiovascular co-morbidities among renal patients. Randomized, controlled trials have shown that normalization of haemoglobin (Hb) with recombinant erythropoietin (rh-Epo) is of no cardiovascular benefit in haemodialysis patients with symptomatic heart failure, ischaemic heart disease, or severe left ventricular dilatation, although suggestive evidence exists for benefits at earlier stages of cardiac disease. Results from large-scale clinical trials are required to clarify the effects of early anaemia correction on mortality and cardiovascular function, as well as appropriate treatment targets in different patient populations. The potential exists for higher Hb levels to extend patient survival through cardioprotective effects.

ACCESSION NUMBER: 2001544119 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11590256
TITLE: Anaemia in chronic renal disease: lessons learned since Seville 1994.
AUTHOR: Parfrey P
CORPORATE SOURCE: Division of Nephrology, Memorial University, St John's, Newfoundland A1B 3V6, Canada.
SOURCE: Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association - European Renal Association, (2001) Vol. 16 Suppl 7, pp. 41-5. Ref: 17
Journal code: 8706402. ISSN: 0931-0509.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20011010
Last Updated on STN: 20020128
Entered Medline: 20020123

L3 ANSWER 5 OF 5 MEDLINE on STN

TI Cardiovascular consequences of renal anaemia and erythropoietin therapy.

AB Cardiovascular disease is the leading cause of increased mortality in patients with renal failure and vigorous attention to cardiovascular risk

factors is therefore required to improve patient outcome. The availability of recombinant human **Epo** has focused the interest on the role of chronic anaemia in the pathogenesis of cardiovascular disease. Severalfold evidence indicates that anaemia can contribute to cardiac volume overload and together with overhydration, fistula flow and the pressure overload secondary to arterial hypertension, it may play a significant role in the development of cardiac hypertrophy. As in the general population left ventricular hypertrophy is a severe adverse risk factor in renal patients. In addition, in the presence of ischaemic **heart disease** anaemia may further worsen cardiac oxygen supply. This dual effect of anaemia probably explains why epidemiological studies have shown that a 1 g/dl decrease in haemoglobin levels is an independent, statistically significant risk factor for the development of cardiac morbidity and mortality. Follow-up examinations have demonstrated that partial correction of anaemia with recombinant **Epo** can improve cardiac oxygen supply and partially reverse pathological changes in left ventricular geometry. However, although partial anaemia correction regularly reduces left ventricular volume, the effects on wall thickness are far less significant. Moreover, in patients with advanced cardiac disease it has recently not been possible to demonstrate that a normalization of haemoglobin levels provides further benefit. It is not unlikely therefore that the development of severe anaemia has to be prevented by early implementation of **Epo** therapy in order to achieve the maximum benefit with respect to the cardiovascular system.

ACCESSION NUMBER: 1999274190 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10344393
TITLE: Cardiovascular consequences of renal anaemia and erythropoietin therapy.
AUTHOR: Eckardt K U
CORPORATE SOURCE: Department of Nephrology and Medical Intensive Care, Charite, Humboldt University, Berlin, Germany.
SOURCE: Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association - European Renal Association, (1999 May) Vol. 14, No. 5, pp. 1317-23. Ref: 64
Journal code: 8706402. ISSN: 0931-0509.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990806
Last Updated on STN: 19990806
Entered Medline: 19990728

=> file biosis

COST IN U.S. DOLLARS	SINCE FILE	TOTAL.
	ENTRY	SESSION
FULL ESTIMATED COST	1.49	1.70

FILE 'BIOSIS' ENTERED AT 09:37:55 ON 04 MAR 2006
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 1 March 2006 (20060301/ED)

```

=> s 11
    6227 EPO
    533892 HEART
    2729600 DISEASE
    248157 HEART DISEASE
          (HEART(W)DISEASE)
L4      111 EPO AND HEART DISEASE

=> s 12
    6227 EPO
    66290 ANEMIA
L5      1590 EPO AND ANEMIA

=> s 13
    6227 EPO
    66290 ANEMIA
    6227 EPO
    533892 HEART
    2729600 DISEASE
    248157 HEART DISEASE
          (HEART(W)DISEASE)
L6      43 L2 AND L1

=> s 16 and PEG
    12980 PEG
L7      0 L6 AND PEG

=> s 16 and glycosylation
    24070 GLYCOSYLATION
L8      0 L6 AND GLYCOSYLATION

=> s 16 and darbepoetin
    302 DARBEPOETIN
L9      0 L6 AND DARBEPOETIN

=> s 16 and human
    6699924 HUMAN
L10     40 L6 AND HUMAN

=> s 110 and (free amino group)
    470724 FREE
    520405 AMINO
    847790 GROUP
    325 FREE AMINO GROUP
          (FREE(W)AMINO(W)GROUP)
L11     0 L10 AND (FREE AMINO GROUP)

=> s 110 and analogs
    50659 ANALOGS
L12     0 L10 AND ANALOGS

=> s 110 and (lower alkoxy PEG group)
    697593 LOWER
    3558 ALKOXY
    12980 PEG
    847790 GROUP
    0 LOWER ALKOXY PEG GROUP
          (LOWER(W)ALKOXY(W)PEG(W)GROUP)
L13     0 L10 AND (LOWER ALKOXY PEG GROUP)

=> d 110 ti abs ibib 1-6

```

TI **Anemia** in chronic heart failure: Can **EPO** reduce deaths?.

AB Many patients with chronic heart failure also have **anemia**, an association that has been increasingly recognized in recent years. Whether treating **anemia** will improve outcomes in patients with heart failure has yet to be determined, however. The decision to use an agent to treat **anemia** in heart failure should be made on a case-by-case basis.

ACCESSION NUMBER: 2005:560788 BIOSIS
DOCUMENT NUMBER: PREV200510336269
TITLE: **Anemia** in chronic heart failure: Can **EPO** reduce deaths?.

AUTHOR(S): Iyengar, Srinivas [Reprint Author]; Abraham, William T.
CORPORATE SOURCE: Ohio State Univ, Div Cardiovasc Med, 473 W 12th Ave, Room 110P DHLRI, Columbus, OH 43210 USA
iyengar-1@medctr.osu.edu

SOURCE: Cleveland Clinic Journal of Medicine, (NOV 2005) Vol. 72, No. 11, pp. 1027-1032.
ISSN: 0891-1150.

DOCUMENT TYPE: Article
General Review; (Literature Review)

LANGUAGE: English

ENTRY DATE: Entered STN: 7 Dec 2005
Last Updated on STN: 7 Dec 2005

L10 ANSWER 2 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

TI Blunted erythropoietin production and defective iron supply for erythropoiesis as major causes of anaemia in patients with chronic heart failure.

AB Aims Anaemia is often observed in patients with chronic heart failure (CHF), and it may be associated with a worse prognosis. Aim of this study was to identify the individual mechanisms of anaemia in CHF patients. Methods and results One hundred and forty-eight consecutive patients with haemoglobin concentration < 13 g/dL (if males) or < 12 g/dL (if females) were enrolled. Factors responsible for anaemia were investigated by evaluating endogenous erythropoietin (**Epo**) production, serum cytokines levels, body iron status, and iron supply for erythropoiesis. Most patients (57%) presented anaemia of chronic disease and among them, 92% showed evidence of a defective endogenous **Epo** production. This was indicated by an observed/predicted log(serum **Epo**) ratio less than 0.8 and/or a defective iron supply for erythropoiesis diagnosed by low transferrin saturation and/or increased value of soluble transferrin receptor. According to regression analysis sex, renal failure, and serum **Epo** were correlated with anaemia. Conclusion According to our study, about half of anaemic CHF patients showed anaemia of chronic disease with blunted endogenous **Epo** production and/or a defective iron supply for erythropoiesis. Determination of the individual mechanisms of anaemia in CHF could justify a rational therapeutic approach to anaemia.

ACCESSION NUMBER: 2005:504068 BIOSIS
DOCUMENT NUMBER: PREV200510277193
TITLE: Blunted erythropoietin production and defective iron supply for erythropoiesis as major causes of anaemia in patients with chronic heart failure.

AUTHOR(S): Opasich, Cristina [Reprint Author]; Cazzola, Mario; Scelsi, Laura; De Feo, Stefania; Bosimini, Enzo; Lagioia, Rocco; Febo, Oreste; Ferrari, Roberto; Fucili, Alessandro; Moratti, Remigio; Tramarin, Roberto; Tavazzi, Luigi

CORPORATE SOURCE: IRCCS, Fdn Salvatore Maugeri, Inst Pavia, Dept Cardiol, Str Ferrara 8, I-27100 Pavia, Italy
copasich@fsm.it

SOURCE: European Heart Journal, (NOV 2005) Vol. 26, No. 21, pp. 2232-2237.

CODEN: EHJODF. ISSN: 0195-668X.

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005

L10 ANSWER 3 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Safety and efficacy of epoetin alfa initiated at 80,000 U once weekly in
anemic cancer patients receiving chemotherapy.
AB Epoetin alfa (**EPO**) 40,000-60,000 U SC once weekly (QW) has been
shown to increase hemoglobin (Hb) by similar to 2 g/dL after 8-12 wks in
anemic patients (pts) with cancer receiving chemotherapy (CT). The
proportion of pts achieving a hematologic response (HR; Hb \geq 12 g/dL
and/or Hb increase \geq 2 g/dL from baseline [BL]) at this dosage was
similar to 70%. It was hypothesized that higher initiation doses of
EPO may result in earlier HR and improved HR rates. This
nonrandomized, open-label, pilot study was designed to investigate the
safety and efficacy of **EPO** 80,000 U SC QW for up to 12 wks in
anemic (Hb \leq 11 g/dL) pts with nonmyeloid malignancies undergoing CT. If Hb
increased to $>$ 13 g/dL, **EPO** was held until Hb \leq 12 g/dL, then dose reduced
by 20,000 U. Dose was similarly reduced if Hb increased $>$ 1.3 g/dL in 2
wks. Primary endpoint was proportion of pts with a major response,
defined as HR. Secondary endpoints included proportion of pts with a
minor response (Hb increase \geq 1 g/dL but $<$ 2 g/dL from BL), time to major
or minor response, Hb over time, and transfusions. Response rates were
independent of RBC transfusion within previous 28 days. Weekly Hb was
analyzed using last value carried forward method to impute missing Hb
values; Hb values within 28 days following an RBC transfusion were
excluded. Sixty-nine pts were enrolled, 46 (67%) of whom completed
through the final study visit. All 69 pts (64% women; mean age 62 y; 83%
ECOG PS 0-1) received \geq 1 dose of study drug and were evaluable for
safety; 68 pts were evaluable for efficacy (modified intent-to-treat
population: \geq 1 post-BL Hb or transfusion value). Most common tumor
types were breast (28%), colorectal (16%), lung (16%), and ovarian (13%).
Mean BL Hb was 10.1 \pm 0.8 g/dL (n=64). Thirty-seven (54%) pts had \geq 1
dose reduction and 33 (48%) pts had \geq 1 dose held. Median time to first
dose reduction or hold was 28 days (n=48) and the mean dose was
approximately 62,600 U/wk. Pts were evaluated for best response, of which
49 (72%) pts achieved a major response and 6 (9%) achieved a minor
response. Major and minor responses were achieved after a median of 6.0
wks (n=49) and 3.0 wks (n=44; includes pts with minor response at best or
minor response followed by major response), respectively. Mean changes in
Hb after 4 wks, 8 wks, and at final value were 1.0 \pm 1.4 g/dL (n=60), 1.7
 \pm 1.4 g/dL (n=62), and 2.0 \pm 1.4 g/dL (n=62), respectively. Six (9%)
pts were transfused at any point during the study; 2 (3%) were transfused
after day 28. Adverse events (Aes) were reported in 65 (94%) pts; most
commonly reported Aes were nausea and neutropenia. Nineteen (28%) pts had
serious Aes. Six (9%) pts had Aes that led to study discontinuation, 4 of
whom died. Five (7%) pts were diagnosed with a total of 6 clinically
relevant thrombotic vascular events (3 deep venous thromboses, 2
pulmonary emboli, 1 cardiac arrest); none was considered related to study
drug. These pilot data showed that this **EPO** dosing regimen
increased Hb by 1.7 g/dL after 8 wks in pts with cancer and anemia
receiving CT. The HR rate of similar to 70% and safety profile observed
with **EPO** 80,000 U SC QW were similar to historical results from
studies of **EPO** 40,000 U SC QW with dose escalation to 60,000 U
SC QW, perhaps related to frequent dose reductions and holds in the
present study or a potential plateau in response at doses greater than
40,000-60,000 U SC QW.

ACCESSION NUMBER: 2005:480293 BIOSIS
DOCUMENT NUMBER: PREV200510266878
TITLE: Safety and efficacy of epoetin alfa initiated at 80,000 U
once weekly in anemic cancer patients receiving

chemotherapy.
AUTHOR(S): Waltzman, Roger J. [Reprint Author]; Braly, Patricia;
Williams, Denise
CORPORATE SOURCE: St Vincents Comprehens Canc Ctr, Dept Med, New York, NY USA
SOURCE: Blood, (NOV 16 2004) Vol. 104, No. 11, Part 2, pp. 143B.
Meeting Info.: 46th Annual Meeting of the
American-Society-of-Hematology. San Diego, CA, USA.
December 04 -07, 2004. Amer Soc Hematol.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005

L10 ANSWER 4 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Erythropoietin mimics ischemic preconditioning.
AB Ischemic preconditioning is a powerful endogenous phenomenon in which
brief periods of a sub-toxic ischemic insult induce robust protection
against future, lengthy, lethal ischemia. The cardioprotective effects of
ischemic preconditioning are manifest in all species studied so far,
including humans. The ability to reproduce the cardioprotective effects
of ischemic preconditioning with pharmacological agents raised the
possibility that a drug may ultimately be introduced into clinical
practice to treat **human** hearts undergoing ischemia/reperfusion.
This chapter focuses on erythropoietin (**Epo**), a drug that has
already been approved for humans and is in current use for the treatment
of **anemia** associated with chronic renal failure, HIV infection,
cancer patients on chemotherapy, and to reduce allogenic blood transfusion
in surgery patients. Several recent studies have suggested that this
cytokine possesses properties far beyond its capacity to produce red blood
cells such as the ability to protect tissues including brain, kidney and
heart against injury caused by ischemia/reperfusion. Cardioprotection
conferred by **Epo** has been shown to be equal in magnitude to that
conferred by ischemic preconditioning. However, the underlying mechanisms
by which **Epo** protects the heart against injury caused by
ischemia remain unknown. (c) 2005 Elsevier Inc. All rights reserved.

ACCESSION NUMBER: 2005:364762 BIOSIS
DOCUMENT NUMBER: PREV200510155541
TITLE: Erythropoietin mimics ischemic preconditioning.
AUTHOR(S): Baker, John E. [Reprint Author]
CORPORATE SOURCE: Med Coll Wisconsin, 8701 Watertown Plank Rd, Milwaukee, WI
53226 USA
jbaker@mcw.edu
SOURCE: Vascular Pharmacology, (APR-MAY 2005) Vol. 42, No. 5-6, pp.
233-241.
ISSN: 1537-1891.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Sep 2005
Last Updated on STN: 14 Sep 2005

L10 ANSWER 5 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Effect of erythropoietin therapy on red cells filterability and left
ventricular mass in predialysis patients.
AB Background. Cardiovascular complications are the leading cause of
mortality in patients with end-stage renal disease. Left ventricular
hypertrophy (LVH) is recognized as an independent risk factor for
cardiovascular morbidity and mortality. At the onset of dialysis, more
than 70% of the patients with chronic kidney disease have
echocardiographic evidence of LVH. **Anemia**, increased red cells
filterability time (RCFT), and blood viscosity are known to induce LVH.

Aim. To evaluate, prospectively, the effects of erythropoietin (**EPO**) therapy for 20 weeks on RCFT and left ventricular mass (LVM). Patients and Methods. Twenty uremic and anemic predialysis patients with creatinine clearance test below 35 mL/min were studied. RCFT test and three-dimensional echocardiography were performed at 0, 10, and 20 weeks. Results. **EPO** therapy for 20 weeks did not adversely affect renal function and did not significantly change the mean blood pressure. It significantly increased the hemoglobin and fibrinogen levels, and decreased RCFT and LVM ($p < .01$). Conclusion. Although correction of **anemia** can contribute to regression of LVM, we speculate that an increasing number of cells with normalized viscoelastic properties and a direct effect of **EPO** on erythrocytes and myocardiocytes, through specific receptors, may also play an important role.

ACCESSION NUMBER: 2005:239011 BIOSIS
 DOCUMENT NUMBER: PREV200510033406
 TITLE: Effect of erythropoietin therapy on red cells filterability and left ventricular mass in predialysis patients.
 AUTHOR(S): Hassan, Kamal [Reprint Author]; Roguin, Nathan; Kaganov, Yan; Hasan, Shadi; Kristal, Batya
 CORPORATE SOURCE: Western Galilee Hosp, Peritoneal Dialysis Unit, Dept Hypertens and Nephrol, POB 21, IL-22100 Nahariya, Israel drkamalh@hotmail.com
 SOURCE: Renal Failure, (2005) Vol. 27, No. 2, pp. 177-182. CODEN: REFAE8. ISSN: 0886-022X.
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 29 Jun 2005
 Last Updated on STN: 29 Jun 2005

L10 ANSWER 6 OF 40 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
 TI The role of **anemia** in the progression of congestive heart failure. Is there a place for erythropoietin and intravenous iron?.

AB **Anemia** is found in about one-third of all cases of congestive heart failure (CHF). The most likely common cause is chronic kidney insufficiency (CKI), which is present in about half of all CHF cases. The CKI is likely to be due to the renal vasoconstriction that often accompanies CHF and can cause long-standing renal ischemia. This reduces the amount of erythropoietin (**EPO**) produced in the kidney and leads to **anemia**. However, **anemia** can occur in CHF without CKI and is likely to be due to excessive cytokine production (for example, tumor necrosis factor-alpha (TNF-alpha) and interleukin-6 (IL-6)), which is common in CHF and can cause reduced **EPO** secretion, interference with **EPO** activity in the bone marrow and reduced iron supply to the bone marrow. The **anemia** itself can worsen cardiac function, both because it causes cardiac stress through tachycardia and increased stroke volume, and because it can cause a reduced renal blood flow and fluid retention, adding further stress to the heart. Long-standing **anemia** of any cause can cause left ventricular hypertrophy (LVH), which can lead to cardiac cell death through apoptosis and worsen the CHF. Therefore, a vicious circle is set up wherein CHF causes **anemia**, and the **anemia** causes more CHF and both damage the kidneys worsening the **anemia** and the CHF further. We have termed this vicious circle the cardio renal **anemia** (CRA) syndrome. Patients with CHF who are anemic are often resistant to all CHF medications resulting in being hospitalized repeatedly. Many studies also demonstrate that these patients die more rapidly than their non-anemic counterparts do. In addition, they have a more rapid deterioration in their renal function and can end up on dialysis. There is now evidence from both uncontrolled and controlled studies that early correction of the CHF **anemia** with subcutaneous **EPO** and intravenous (i.v.) iron improves shortness of breath and fatigue, cardiac function, renal function and exercise capability, dramatically reducing the need for hospitalization. For these

reasons, it is not surprising that quality of life has also been shown to improve. As both CHF and end-stage renal disease (ESRD) are rapidly increasing, the possibility that these twin conditions can be improved by the adequate treatment of **anemia** offers new hope for slowing the progression of both conditions.

ACCESSION NUMBER: 2005:119703 BIOSIS
DOCUMENT NUMBER: PREV200500118141
TITLE: The role of **anemia** in the progression of congestive heart failure. Is there a place for erythropoietin and intravenous iron?
AUTHOR(S): Silverberg, Donald S. [Reprint Author]; Wexler, Dov; Iaina, Adrian
CORPORATE SOURCE: Dept Nephrol, Tel Aviv Sourasky Med Ctr, Weizman 6; IL-64239, Tel Aviv, Israel
donald@netvision.net.il
SOURCE: JN Journal of Nephrology, (November 2004) Vol. 17, No. 6, pp. 749-761. print.
ISSN: 1121-8428.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Mar 2005
Last Updated on STN: 23 Mar 2005

=> d his

(FILE 'HOME' ENTERED AT 09:37:18 ON 04 MAR 2006)

FILE 'MEDLINE' ENTERED AT 09:37:26 ON 04 MAR 2006

L1 19 S EPO AND HEART DISEASE
L2 1544 S EPO AND ANEMIA
L3 5 S L2 AND L1

FILE 'BIOSIS' ENTERED AT 09:37:55 ON 04 MAR 2006

L4 111 S L1
L5 1590 S L2
L6 43 S L3
L7 0 S L6 AND PEG
L8 0 S L6 AND GLYCOSYLATION
L9 0 S L6 AND DARBEPOETIN
L10 40 S L6 AND HUMAN
L11 0 S L10 AND (FREE AMINO GROUP)
L12 0 S L10 AND ANALOGS
L13 0 S L10 AND (LOWER ALKOXY PEG GROUP)

=> file wpids

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	15.81	17.51

FILE 'WPIDS' ENTERED AT 09:39:57 ON 04 MAR 2006
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FILE LAST UPDATED: 2 MAR 2006 <20060302/UP>
MOST RECENT DERWENT UPDATE: 200615 <200615/DW>
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http://www.stn-international.de/stndatabases/details/ipc_reform.html and
<http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf> <<<

=> s 11

709 EPO
42267 HEART
118834 DISEASE
6160 HEART DISEASE
(HEART(W) DISEASE)
L14 11 EPO AND HEART DISEASE

=> s 16

709 EPO
4647 ANEMIA
709 EPO
42267 HEART
118834 DISEASE
6160 HEART DISEASE
(HEART(W) DISEASE)

L15 2 L2 AND L1

=> d 115 ti abs ibib tot

L15 ANSWER 1 OF 2 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
TI Using erythropoietin to stimulate endothelial precursor cells, useful e.g.
for treating diabetes and inflammation, also for preparing transplants and
coating prostheses.
AN 2004-170502 [17] WPIDS
AB DE 10234192 A UPAB: 20040310
NOVELTY - Use of erythropoietin (**EPO**) and/or its derivatives for
stimulating, in endothelial precursor cells (EPC), any of physiological
mobilization; proliferation; differentiation to endothelial cells and/or
migration, in the direction of an angiogenic or vasculogenic stimulus.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the
following:
(1) use of **EPO** and/or its derivatives for stimulating
formation of endothelial cells (EC) and/or for vasculogenesis;
(2) use of **EPO** to produce a transplantable EC product;
(3) use of **EPO** for treating a tissue or organ transplant;
(4) use of **EPO** for pretreatment of tissue or organ
transplants;
(5) use of **EPO** to prepare implantable or transplantable,
cell-containing in vitro organ or tissue systems;
(6) use of **EPO** for coating vascular prostheses or heart
valves; and

(7) pharmaceutical composition containing (a) **EPO**, its derivatives, analogs, modifications or muteins and (b) at least one of vascular endothelial growth factor, PIGF (not defined), granulocyte-macrophage colony-stimulating factor and/or a hydroxymethyl glutarate-CoA reductase inhibitor.

ACTIVITY - Antilipemic; Antidiabetic; Antiinflammatory; Antiarteriosclerosis; Cardiant; Vasotropic; Antianginal; Gynecological; Hypotensive; Nephrotropic. No details of tests for any of these activities are given.

MECHANISM OF ACTION - Treatment with **EPO** increases adhesion of EPC and stimulates formation of endothelial tissue and blood vessels.

USE - **EPO** is used for treatment of human and animal diseases associated with dysfunction of EPC and/or endothelial cells (EC), particularly where formation of endothelial tissue or blood vessels is affected, specifically hypercholesterolemia; diabetes mellitus; chronic inflammation; reticulo-endotheliosis; atherosclerosis; coronary heart disease; myocardial ischemia; angina pectoris; age-related cardiovascular disease; ischemic disease of the extremities; pre-eclampsia; Raynaud disease; pregnancy-induced hypertension; renal insufficiency (especially end-stage disease) and their sequelae. It can also be used to prepare transplanatable EC compositions; to pretreat tissue or organ transplants; to prepare implantable/transplantable cell-containing in vitro organ or tissue systems, and to coat vascular prostheses or heart valves (all these uses are claimed).

Dwg.0/3

ACCESSION NUMBER: 2004-170502 [17] WPIDS
 DOC. NO. CPI: C2004-067648
 TITLE: Using erythropoietin to stimulate endothelial precursor cells, useful e.g. for treating diabetes and inflammation, also for preparing transplants and coating prostheses.
 DERWENT CLASS: B04 D16 D22
 INVENTOR(S): BAHLMANN, F H; HALLER, H
 PATENT ASSIGNEE(S): (BAHL-I) BAHLMANN F H; (EPOP-N) EPOPLUS GMBH & CO KG; (HALL-I) HALLER H
 COUNTRY COUNT: 106
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
DE 10234192	A1	20040212	(200417)*		17
WO 2004012759	A2	20040212	(200417)	GE	
RW:	AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW				
W:	AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW				
AU 2003255290	A1	20040223	(200453)		
EP 1526867	A2	20050504	(200530)	GE	
R:	AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR				
NO 2005001002	A	20050418	(200535)		
BR 2003012981	A	20050614	(200541)		
KR 2005026513	A	20050315	(200557)		
US 2005272634	A1	20051208	(200581)		
JP 2006503001	W	20060126	(200609)		51
CN 1681526	A	20051012	(200612)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
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DE 10234192	A1	DE 2002-10234192	20020726
WO 2004012759	A2	WO 2003-EP8229	20030725
AU 2003255290	A1	AU 2003-255290	20030725
EP 1526867	A2	EP 2003-766302	20030725
		WO 2003-EP8229	20030725
NO 2005001002	A	WO 2003-EP8229	20030725
		NO 2005-1002	20050224
BR 2003012981	A	BR 2003-12981	20030725
		WO 2003-EP8229	20030725
KR 2005026513	A	KR 2005-701341	20050125
US 2005272634	A1	WO 2003-EP8229	20030725
		US 2005-522426	20050325
JP 2006503001	W	WO 2003-EP8229	20030725
		JP 2004-525322	20030725
CN 1681526	A	CN 2003-822116	20030725

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003255290	A1 Based on	WO 2004012759
EP 1526867	A2 Based on	WO 2004012759
BR 2003012981	A Based on	WO 2004012759
JP 2006503001	W Based on	WO 2004012759

PRIORITY APPLN. INFO: DE 2002-10234192 20020726

L15 ANSWER 2 OF 2 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

TI Novel isolated nucleic acid comprising a region that codes for first regulatory module operably linked to region that codes for insulator, which is linked to a region that codes for second different regulatory module.

AN 2003-493403 [46] WPIDS

AB WO2003046133 A UPAB: 20030719

NOVELTY - Isolated nucleic acids which comprise several regulatory module combinations, are new, e.g. an isolated nucleic acid (Ia) comprising a region that codes for a first regulatory module (R1) operably linked to a region that codes for an insulator, the region that codes for insulator being operably linked to a region that codes for second regulatory module (R2) that is different from region that codes for R1.

DETAILED DESCRIPTION - Isolated nucleic acids which comprise several regulatory module combinations, are provided, e.g.

(a) an isolated nucleic acid (Ia) comprising a region that codes for a first regulatory module (R1) operably linked to a region that codes for an insulator (I);

(b) an isolated nucleic acid (Ib) comprising a region that encodes a first reporter gene operably linked to a region that codes for R1 which is linked to a region that codes for an insulator that is operably linked to a region that codes for R2 that is different from R1;

(c) an isolated nucleic acid (Ic) comprising coding region for first fluorescent protein operably linked to first promoter sequence which is linked to enhancer sequence that is linked to cHS4 insulator sequence that is operably linked to second enhancer sequence that is different from the first enhancer sequence; and

(d) an isolated nucleic acid (Id) comprising a region that codes for (R1) operably linked to a region that encodes first reporter gene which is operably linked to a region that encodes an insulator, where the insulator is operably linked to a region that codes for R2 that is different from R1.

INDEPENDENT CLAIMS are also included for the following:

(1) vectors comprising (Ia) labelled as (II), (Ib) labelled as (V), (Ic) and (Id) not labelled;

- (2) an isolated, genetically modified cell (III) comprising (II);
- (3) a cell (IV) modified to contain (Ia) or (II);
- (4) constructing a regulatory sequence, involves operably linking a first sequence comprising the coding sequence for R1 with a second sequence comprising the coding sequence for an insulator, operably linking the second sequence with a third sequence comprising the coding sequence for R2, where the first and third sequences code for different regulatory modules;
- (5) a library of (I);
- (6) an isolated genetically modified cell (VI) comprising (V);
- (7) an isolated nucleic acid (VII) which comprises a fully defined BTEB hypoxia enhancer, CG Orphan H1 enhancer, collagen type 4-A1 (Col4A1) hypoxia enhancer, Deltex hypoxia enhancer, GRAP hypoxia enhancer, PROC hypoxia enhancer, relA hypoxia enhancer, or laminin binding protein (LBP)-32 hypoxia enhancer sequence as given in the specification;
- (8) an isolated nucleic acid (VIII) comprising an expression system which is sensitive to hypoxia, comprising an enhancer sequence operably linked to a promoter and a nucleotide sequence to be expressed where the presence of the enhancer sequence results in increased transcription, under hypoxic conditions, of the nucleotide sequence to be expressed;
- (9) an isolated nucleic acid (IX) comprising any one of enhancer sequences as described above, or other enhancer sequences such as human monooxygenase 1 (HMOX1) hypoxia enhancer, ELL hypoxia enhancer, APAF1 hypoxia enhancer, P53R2 hypoxia enhancer etc., or its fragment or variant, where the fragment or variant enhancer activity that increases transcription under hypoxic conditions;
- (10) a vector (X) comprising (IX);
- (11) a genetically modified cell (XI) comprising (X);
- (12) increasing expression of a nucleotide sequence in a cell under hypoxic conditions involves contacting the cell with a compound which increases the expression of nucleotide sequence to be expressed in a cell, where the compound is identified using (VIII);
- (13) a molecular device which is controlled by hypoxia-related stress, comprising (VIII);
- (14) determining a change in oxygen levels of a maintained cell (method fully defined in the specification);
- (15) inserting a reporter gene into (VIII) involves operably linking the reporter gene to the nucleic acid;
- (16) identifying an agent that regulates the expression of a gene in response to hypoxia;
- (17) identifying (M1) at least one compound that interacts with a test pathway (method fully disclosed in the specification); and
- (18) altering (M2) a protein-protein interaction in a test pathway or affecting compound-protein interaction in a test pathway, (method fully disclosed in the specification).

ACTIVITY - Vasotropic; Cytostatic; Cerebroprotective; Cardiant; Antianemic.

MECHANISM OF ACTION - Regulates gene expression in response to hypoxia. No supporting data is given.

USE - (II) and (VI) are useful for transfecting a cell. (V) is useful for altering the expression of a reporter gene in a cell. (VIII) is useful for identifying compounds which affect response to hypoxia, for inhibiting gene expression, for isolating nucleic acid binding proteins, for constructing a regulatory sequence, altering the expression of nucleotide sequence to be expressed in cell, and identifying the compound which increases the expression of a nucleotide sequence to be expressed (claimed). The compounds identified by above mentioned methods are useful for treating hypoxia-related disorders, e.g., ischemic heart disease, cancer, stroke, chronic lung disease, congestive heart failure, anemia, etc.

Dwg.0/9

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TITLE: Novel isolated nucleic acid comprising a region that codes for first regulatory module operably linked to region that codes for insulator, which is linked to a region that codes for second different regulatory module.

DERWENT CLASS: B04 D16

INVENTOR(S): ERIVES, A J

PATENT ASSIGNEE(S): (ERIV-I) ERIVES A J; (AUIL-N) AUILIX BIOPHARMA INC

COUNTRY COUNT: 102

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003046133	A2	20030605	(200346)*	EN	88
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM ZW					
US 2003134263	A1	20030717	(200348)		
AU 2002352852	A1	20030610	(200419)		
AU 2002352852	A8	20051013	(200611)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003046133	A2	WO 2002-US37412	20021120
US 2003134263	A1	US 2001-989993	20011121
AU 2002352852	A1	AU 2002-352852	20021120
AU 2002352852	A8	AU 2002-352852	20021120

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002352852	A1 Based on	WO 2003046133
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PRIORITY APPLN. INFO: US 2001-989993 20011121